# Cleaved fibrinogen products bind

# TLR4:LY96

Granucci, F., Shamovsky, V., Zanoni, I.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.



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# Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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## Literature references

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Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

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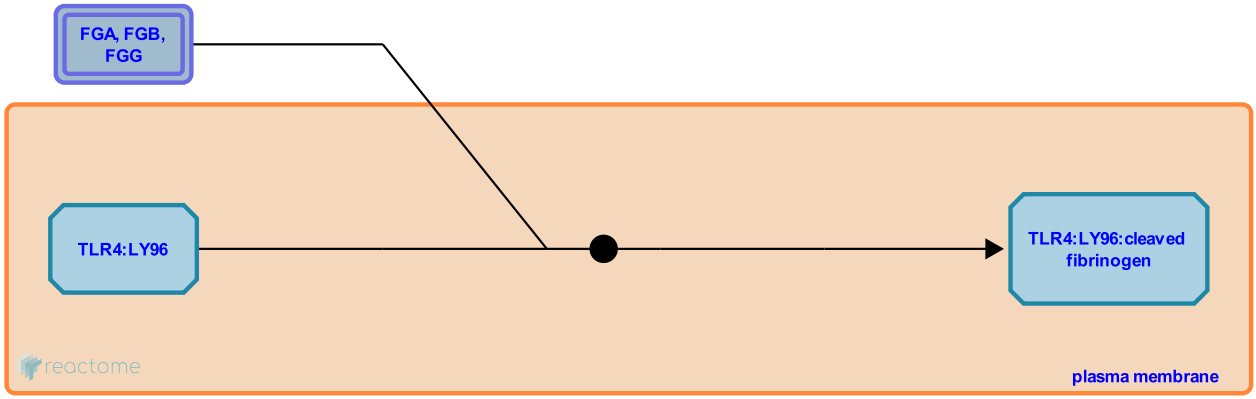
## Cleaved fibrinogen products bind TLR4:LY96

**Stable identifier:** R-HSA-8870678

**Type:** binding

**Compartments:** plasma membrane




Fibrinogen, in addition to its role in coagulation, is also an acute phase protein of inflammation which can induce a cytokine production acting as an endogenous ligand for toll-like receptor 4 (TLR4) expressed on cells including macrophages and airway epithelial cells (Millien VO et al., 2013; Kuhns DB et al., 2007; Smiley ST et al., 2001). In human macrophages fibrinogen stimulated interleukin IL6 expression and extracellular signal-related kinase (ERK) phosphorylation ((Hodgkinson CP et al., 2008). In human embryonic kidney 293 (HEK293)-CD14-MD2 cells expressing TLR4, fibrinogen induced robust phosphorylation of ERK1, p38alpha and JNK and activated transcription factors NFkappaB, Elk1 and AP1 (Hodgkinson CP et al., 2008). Moreover, proteinases, such as thrombin, can cleave fibrinogen. In mice, exposure to endogenous or exogenous proteinases lead to hyperactivation of an antifungal pathway and lead to allergic airway inflammation through activation of TLR4-dependent signaling pathway by fibrinogen cleaved products (Millien VO et al., 2013)

## Literature references

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## Editions

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